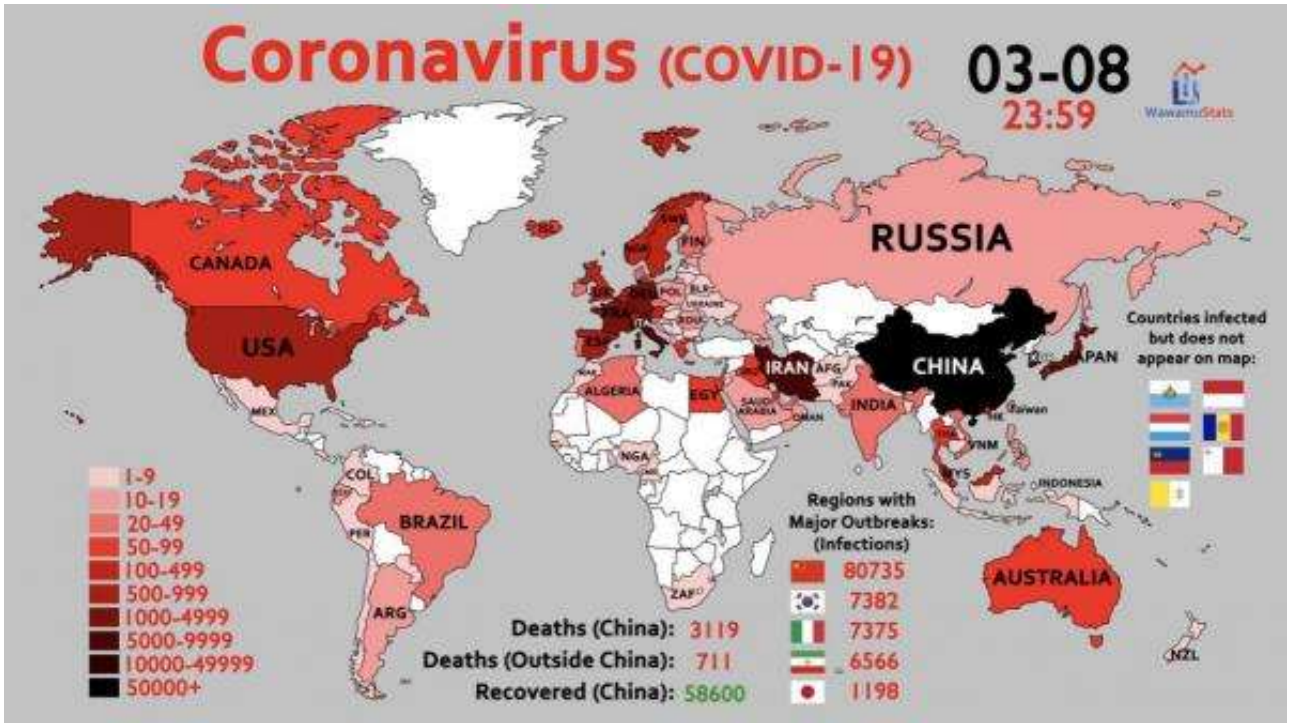
Nature magazine leaked the origin of COVID-19 - Temple of Literature - World Wide Blog - World Wide Reader Network (computer version)

According to the website of the Ministry of Foreign Affairs of China on March 11, Foreign Ministry spokesman Geng Shuang hosted a regular press conference that day and answered reporters' questions about the COVID-19 epidemic. The reporter asked, "Last week, your colleague said in response to the reporter's question that the COVID-19 epidemic first appeared in China, but it did not necessarily originate in China. So do you know which country the virus originated from?" Geng replied: "Regarding the source of the virus, this is a scientific issue, and we need to listen to scientific and professional opinions. We hope that the relevant parties will not take this opportunity to make articles, nor will they use this to stigmatize other countries, which is not conducive to the international community's concerted efforts to fight the epidemic.

Wuhan pneumonia is from Wuhan, and the city also has a top P4 virus research institute. At the beginning of the epidemic, public opinion pointed all the finger at Shi Zhengli, a virus expert of the institute. A large number of her Chinese and English articles on coronavirus, government awards, and even recruitment advertisements were dug up. In the panic, the media in Beijing desperately smeared the primitive host bat, and the intermediate host relied on the pangolin, fruit civet, snake, etc. Real-life dummings, such as Gao Fu, Chen Quanjiao and Huang Yanling, were all pushed to the headlines by the media. In the war of blame and blame, the Communist Party of China panicked and did not even let Mars and the moon go. The recent blame is of course the United States. The first

"unexplained case of infection" found in February is said to be the CDC in the United States announcing that "the first pneumonia case came from the United States", which has become the laughing stock of overseas Chinese news.



But most Chinese people are not so forgetful. Some people found out the media report: "The first confirmed case of "patient zero" in the United States is a Chinese man in his fifties. When he flew from Wuhan to the United States through the passage of Los Angeles International Airport on January 15, he already had symptoms of cough and mild fever. On the 16th, he was diagnosed with novel coronavirus pneumonia. On January 19, because he had had fever and cough for 4 days, and he did have a history of living in Wuhan, so he came to I went to the emergency clinic in Snohomish County, Washington. The second COVID-19 patient in the United States is a white woman in her sixties. She traveled to Wuhan in late December and entered the country through Chicago Airport, and was later diagnosed. Both of them had brief but very close contact with a large number of people at the airport without exception.

However, I agree with Geng Shuang's previous description: "The source

of the virus is a scientific problem, and it is necessary to listen to scientific and professional opinions." Is the United States involved in this novel coronavirus? The answer is very simple. Just look for scientific articles. Considering that most Chinese people don't know English, don't know the major, and may not be able to climb over the wall, I will ask relevant experts to help find two classic articles, explain and partially translate them into Chinese, and look at the source of the virus. I hope Geng Shuang and the audience can understand it.

The world's top scientific magazine Nature and its sub-journal Natural Medicine are rigorous and treasured journals for countless scientific researchers. Among tens of thousands of magazines, its relatively authoritative, objective and impartial position has been seen for a long time. Chinese scientists have also been publishing papers in journals such as Nature as a certificate of honor and national awards. Shi Zhengli and Ge Xingyi of Wuhan Institute of Virology and others were published in Nature magazine https://www.nature.com/articles/nature12711 on October 30, 2013, and in Nature Medicine on November 9, 2015. Important articles on novel coronavirus have been published in the magazine https://www.nature.com/articles/nm.3985. Anyone can access the website of these two articles without restrictions.

Let's take a look at the 2013 Nature magazine, which was completed by Ge Xingyi and other three people (noted as *). As usual, the person in charge is at the end, Shi Zhengli, the corresponding author. Translated into Chinese as: "Isolate and identify Sas-like bat coronavirus, which uses ACE2 receptor". Note that the current epidemic of Wuhan pneumonia virus directly attacks humans, which is the ACE2 receptor. In the abstract, the article claims that bats come from Yunnan, while the applied ACE2 comes from humans. In the method, the analysis of recombination viruses is mentioned, and it becomes a subheading. Although there are collaborators, the Wuhan virus accounts for more than 90% of the total work, especially Shi Zhengli, the top three and the

last responsible.

nature.com

LETTER

doi:10.1038/nature12711

Isolation and characterization of a bat SARS-like coronavirus that uses the ACE2 receptor

Xing-Yi Ge[1]*, Jia-Lu Li[1]*, Xing-Lou Yang[2]*, Aleksei A. Chmura[2], Guangjian Zhu[2], Jonathan H. Epstein[2], Jonna K. Mazet[3], Ben Hu[1], Wei Zhang[1], Cheng Peng[2], Yu-Ji Zhang[1], Chu-Ming Luo[1], Bing Tan[1], Ning Wang[1], Yan Zhu[1], Gary Crameri[4], Shu-Yi Zhang[5], Lin-Fa Wang[4,5], Peter Daszak[2] & Zheng-Li Shi[1]

The 2002–3 pandemic caused by severe acute respiratory syndrome coronavirus (SARS-CoV) was one of the most significant public health events in recent history[1]. An ongoing outbreak of Middle East respiratory syndrome coronavirus[2] suggests that this group of viruses remains a key threat and that their distribution is wider than previously recognized. Although bats have been suggested to be the natural reservoirs of both viruses[3–5], attempts to isolate the progenitor virus of SARS-CoV from bats have been unsuccessful. Diverse SARS-like coronaviruses (SL-CoVs) have now been reported from bats in China, Europe and Africa[3–8], but none is considered a direct progenitor of SARS-CoV because of their phylogenetic disparity from this virus and the inability of their spike proteins to use the SARS-CoV cellular receptor molecule, the human angiotensin converting enzyme II (ACE2)[9,10]. Here we report whole-genome sequences of two novel bat coronaviruses from Chinese horseshoe bats (family: Rhinolophidae) in Yunnan, China: RsSHC014 and Rs3367. These viruses are far more closely related to SARS-CoV than any previously identified bat coronaviruses, particularly in the receptor binding domain of the spike protein. Most importantly, we report the first recorded isolation of a live SL-CoV (bat SL-CoV-WIV1) from bat faecal samples in Vero E6 cells, which has typical coronavirus morphology, 99.9% sequence identity to Rs3367 and uses ACE2 from humans, civets and Chinese horseshoe bats for cell entry. Preliminary *in vitro* testing indicates that WIV1 also has a broad species tropism. Our results provide the strongest evidence to date that Chinese horseshoe bats are natural reservoirs of SARS-CoV, and that intermediate hosts may not be necessary for direct human infection by some bat SL-CoVs. They also highlight the importance of pathogen-discovery programs targeting high-risk wildlife groups in emerging disease hotspots as a strategy for pandemic preparedness.

The 2002–3 pandemic of SARS[1] and the ongoing emergence of the Middle East respiratory syndrome coronavirus (MERS-CoV)[2] demonstrate that CoVs are a significant public health threat. SARS-CoV was shown to use the human ACE2 molecule as its entry receptor, and this is considered a hallmark of its cross-species transmissibility[11]. The receptor binding domain (RBD) located in the amino-terminal region (amino acids 318–510) of the SARS-CoV spike (S) protein is directly involved in binding to ACE2 (ref. 12). However, despite phylogenetic evidence that SARS-CoV evolved from bat SL-CoVs, all previously identified SL-CoVs have major sequence differences from SARS-CoV in the RBD of their S proteins, including one or two deletions[8,9]. Replacing the RBD of one SL-CoV S protein with SARS-CoV S conferred the ability to use human ACE2 and replicate efficiently in mice[9,13]. However, to date, no SL-CoVs have been isolated from bats, and no wild-type SL-CoV of bat origin has been shown to use ACE2.

We conducted a 12-month longitudinal survey (April 2011–September 2012) of SL-CoVs in a colony of *Rhinolophus sinicus* at a single location in Kunming, Yunnan Province, China (Extended Data Table 1). A total of 117 anal swabs or faecal samples were collected from individual bats using a previously published method[3,14]. A one-step reverse transcription (RT)-nested PCR was conducted to amplify the RNA-dependent RNA polymerase (RdRP) motifs A and C, which are conserved among alphacoronaviruses and betacoronaviruses[15].

Twenty-seven of the 117 samples (23%) were classed as positive by PCR and subsequently confirmed by sequencing. The species origin of all positive samples was confirmed to be *R. sinicus* by cytochrome b sequence analysis, as described previously[16]. A higher prevalence was observed in samples collected in October (30% in 2011 and 48.7% in 2012) than those in April (7.1% in 2011) or May (7.4% in 2012) (Extended Data Table 1). Analysis of the S protein RBD sequences indicated the presence of seven different strains of SL-CoVs (Fig. 1a and Extended Data Figs 1 and 2). In addition to RBD sequences, which closely matched previously described SL-CoVs (Rs672, Rf1 and HKU3)[3,8,17,18], two novel strains (designated SL-CoV RsSHC014 and Rs3367) were discovered. Their full-length genome sequences were determined, and both were found to be 29,787 base pairs in size (excluding the poly(A) tail). The overall nucleotide sequence identity of these two genomes with human SARS-CoV (Tor2 strain) is 95%, higher than that observed previously for bat SL-CoVs in China (88–92%)[3,8,17,18] or Europe (76%)[6] (Extended Data Table 2 and Extended Data Figs 3 and 4). Higher sequence identities were observed at the protein level between these new SL-CoVs and SARS-CoVs (Extended Data Tables 3 and 4). To understand the evolutionary origin of these two novel SL-CoV strains, we conducted recombination analysis with the Recombination Detection Program 4.0 package[19] using available genome sequences of bat SL-CoV strains (Rf1, Rp3, Rs672, Rm1, HKU3 and BM48-31) and human and civet representative SARS-CoV strains (BJ01, SZ3, Tor2 and GZ02). Three breakpoints were detected with strong P values ($<10^{-20}$) and supported by similarity plot and bootscan analysis (Extended Data Fig. 5a, b). Breakpoints were located at nucleotides 20,827, 26,553 and 28,685 in the Rs3367 (and RsSHC014) genome, and generated recombination fragments covering nucleotides 20,827–26,533 (5,727 nucleotides) (including partial open reading frame (ORF) 1b, full-length S, ORF3, E and partial M gene) and nucleotides 26,534–28,685 (2,133 nucleotides) (including partial ORF M, full-length ORF6, ORF7, ORF8 and partial N gene). Phylogenetic analysis using the major and minor parental regions suggested that Rs3367, or RsSHC014, is the descendent of a recombination of lineages that ultimately lead to SARS-CoV and SL-CoV Rs672 (Fig. 1b).

The most notable sequence differences between these two new SL-CoVs and previously identified SL-CoVs is in the RBD regions of their S proteins. First, they have higher amino acid sequence identity to SARS-CoV (85% and 96% for RsSHC014 and Rs3367, respectively). Second, there are no deletions and they have perfect sequence alignment with the SARS-CoV RBD region (Extended Data Figs 1 and 2). Structural

[1]Center for Emerging Infectious Diseases, State Key Laboratory of Virology, Wuhan Institute of Virology of the Chinese Academy of Sciences, Wuhan 430071, China. [2]EcoHealth Alliance, New York, New York 10001, USA. [3]One Health Institute, School of Veterinary Medicine, University of California, Davis, California 95616, USA. [4]CSIRO Australian Animal Health Laboratory, Geelong, Victoria 3220, Australia. [5]College of Life Sciences, East China Normal University, Shanghai 200062, China. [6]Emerging Infectious Diseases Program, Duke-NUS Graduate Medical School, Singapore 169857.
*These authors contributed equally to this work.

©2013 Macmillan Publishers Limited. All rights reserved

Isolation and characterization of a bat SARS-like coronavirus that uses the ACE2 receptor

Xing-Yi Ge*, Jia-Lu Li1 *, Xing-Lou Yang1 *, Aleksei A. Chmura2, Guangjian Zhu2, Jonathan H. Epstein2, Jonna K. Mazet3, Ben Hu1, Wei Zhang1, Cheng Peng1, Yu-Ji Zhang1, Chu-Ming Luo1, Bing Tan1, Ning Wang1 , Yan Zhu1, Gary Crameri4, Shu-Yi Zhang5, Lin-Fa Wang4,6, Peter Daszak2 & Zheng-Li Shi

Undoubtedly, in 2013, Shi Zhengli achieved great success in this major, and also attracted many peers as collaborators to continue her research on the novel coronavirus. The reason is very simple. Shi Zhengli has bat specimens and primitive biological materials, but there are technologies abroad. This phenomenon has been very common in the Chinese medical biology community for more than 20 years. The second article was published in the journal Natural Medicine (see above). Many universities in the United States are the main contributors, while Shi Zhengli and Ge Xingyi are only supporting roles or collaborators, supplying basic biological materials, and also participating in some of the work. The article also has a detailed record of the author's division of labor. Many people are unwilling to mention the first article in the Nature magazine, but only this article in Natural Medicine. In this way, it seems that most of the viruses are caused by Americans, although the title of the article is to warn of the dangers of COVID-19. However, the first article in Nature magazine is the basis of the second article, and the second article is a follow-up report of the "source of the virus". Therefore, the first article cited in the second article of Natural Medicine is Shi Zhengli's article in 2013 "Isolated and identified by Sas-like bat coronavirus, which uses ACE2 receptor".

nature.com

nature medicine

A SARS-like cluster of circulating bat coronaviruses shows potential for human emergence

Vineet D Menachery[1], Boyd L Yount Jr[1], Kari Debbink[1,2], Sudhakar Agnihothram[3], Lisa E Gralinski[1], Jessica A Plante[1], Rachel L Graham[1], Trevor Scobey[1], Xing-Yi Ge[4], Eric F Donaldson[1], Scott H Randell[5,6], Antonio Lanzavecchia[7], Wayne A Marasco[8,9], Zhengli-Li Shi[4] & Ralph S Baric[1,2]

© 2015 Nature America, Inc. All rights reserved.

The emergence of severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome (MERS)-CoV underscores the threat of cross-species transmission events leading to outbreaks in humans. Here we examine the disease potential of a SARS-like virus, SHC014-CoV, which is currently circulating in Chinese horseshoe bat populations[1]. Using the SARS-CoV reverse genetics system[2], we generated and characterized a chimeric virus expressing the spike of bat coronavirus SHC014 in a mouse-adapted SARS-CoV backbone. The results indicate that group 2b viruses encoding the SHC014 spike in a wild-type backbone can efficiently use multiple orthologs of the SARS receptor human angiotensin converting enzyme II (ACE2), replicate efficiently in primary human airway cells and achieve *in vitro* titers equivalent to epidemic strains of SARS-CoV. Additionally, *in vivo* experiments demonstrate replication of the chimeric virus in mouse lung with notable pathogenesis. Evaluation of available SARS-based immune-therapeutic and prophylactic modalities revealed poor efficacy; both monoclonal antibody and vaccine approaches failed to neutralize and protect from infection with CoVs using the novel spike protein. On the basis of these findings, we synthetically re-derived an infectious full-length SHC014 recombinant virus and demonstrate robust viral replication both *in vitro* and *in vivo*. Our work suggests a potential risk of SARS-CoV re-emergence from viruses currently circulating in bat populations.

The emergence of SARS-CoV heralded a new era in the cross-species transmission of severe respiratory illness with globalization leading to rapid spread around the world and massive economic impact[3,4]. Since then, several strains—including influenza A strains H5N1, H1N1 and H7N9 and MERS-CoV—have emerged from animal populations, causing considerable disease, mortality and economic hardship for the afflicted regions[5]. Although public health measures were able to stop the SARS-CoV outbreak[4], recent metagenomics studies have identified sequences of closely related SARS-like viruses circulating in Chinese bat populations that may pose a future threat[1,6]. However, sequence data alone provides minimal insights to identify and prepare for future prepandemic viruses. Therefore, to examine the emergence potential (that is, the potential to infect humans) of circulating bat CoVs, we built a chimeric virus encoding a novel, zoonotic CoV spike protein—from the RsSHC014-CoV sequence that was isolated from Chinese horseshoe bats[1]—in the context of the SARS-CoV mouse-adapted backbone. The hybrid virus allowed us to evaluate the ability of the novel spike protein to cause disease independently of other necessary adaptive mutations in its natural backbone. Using this approach, we characterized CoV infection mediated by the SHC014 spike protein in primary human airway cells and *in vivo*, and tested the efficacy of available immune therapeutics against SHC014-CoV. Together, the strategy translates metagenomics data to help predict and prepare for future emergent viruses.

The sequences of SHC014 and the related RsWIV1-CoV show that these CoVs are the closest relatives to the epidemic SARS-CoV strains (**Fig. 1a,b**); however, there are important differences in the 14 residues that bind human ACE2, the receptor for SARS-CoV, including the five that are critical for host range: Y442, L472, N479, T487 and Y491 (ref. 7). In WIV1, three of these residues vary from the epidemic SARS-CoV Urbani strain, but they were not expected to alter binding to ACE2 (**Supplementary Fig. 1a,b** and **Supplementary Table 1**). This fact is confirmed by both pseudotyping experiments that measured the ability of lentiviruses encoding WIV1 spike proteins to enter cells expressing human ACE2 (**Supplementary Fig. 1**) and by *in vitro* replication assays of WIV1-CoV (ref. 1). In contrast, 7 of 14 ACE2-interaction residues in SHC014 are different from those in SARS-CoV, including all five residues critical for host range (**Supplementary Fig. 1c** and **Supplementary Table 1**). These changes, coupled with

[1]Department of Epidemiology, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA. [2]Department of Microbiology and Immunology, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA. [3]National Center for Toxicological Research, Food and Drug Administration, Jefferson, Arkansas, USA. [4]Key Laboratory of Special Pathogens and Biosafety, Wuhan Institute of Virology, Chinese Academy of Sciences, Wuhan, China. [5]Department of Cell Biology and Physiology, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA. [6]Cystic Fibrosis Center, Marsico Lung Institute, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA. [7]Institute for Research in Biomedicine, Bellinzona Institute of Microbiology, Zurich, Switzerland. [8]Department of Cancer Immunology and AIDS, Dana-Farber Cancer Institute, Harvard Medical School, Boston, Massachusetts, USA. [9]Department of Medicine, Harvard Medical School, Boston, Massachusetts, USA. Correspondence should be addressed to R.S.B. (rbaric@email.unc.edu) or V.D.M. (vineet@email.unc.edu).

Received 12 June; accepted 8 October; published online 9 November 2015; corrected online 20 November 2015 (details online); doi:10.1038/nm.3985

Original text of Nature's official website:
https://www.nature.com/articles/nm.3985

A SARS-like cluster of circulating bat coronaviruses shows potential for human emergence

1. Department of Epidemiology, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina , USA. 2. Department of Microbiology and Immunology, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA. 3. National Center for Toxicological Research, Food and Drug Administration, Jefferson, Arkansas, USA. 4. Key Laboratory of Special Pathogens and Biosafety, Wuhan Institute of Virology, Chinese Academy of S ciences, Wuhan, China. 5. Department of Cell Biology and Physiology, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA. 6. Cystic Fibrosis Center, Marsico Lung Institute, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA. 7. Institute for Research in Biomedicine, Bellinzona Institute of Microbiology, Zurich, Switzerland. 8. Department of Cancer Immunology and AIDS, Dana-Farber Cancer Institute, Harvard Medical School, Bost On, Massachusetts, USA. 9. Department of Medicine, Harvard Medical School, Boston, Massachusetts, USA. Received 12 June; accepted 8 October; published online 9 November 2015; corrected online 20 November 2015 (details online); doi:10.1038/nm.3985

The following sections are excerpted, which can clearly see the origin, harm and specific contributions of the virus in Wuhan.

We consider viruses with the **SHC014 spike a potential threat due to their ability to replicate in primary human airway cultures, the best available model for human disease. In addition, the observed**

pathogenesis in mice indicates a capacity for SHC014-containing viruses to cause disease in mammalian models, without RBD adaptation.

Replication in the human respiratory tract and the harm of the virus.

……

Together, the data confirms the ability of viruses with the SHC014 spike to infect human airway cells and underscore the potential threat of cross-species transmission of SHC014-CoV.

The virus may cause cross-species infection, directly threatening human beings.

……

Overall, our approach has used metagenomics data to identify a potential threat posed by the circula ting bat SARS-like CoV SHC014. Because of the ability of chimeric SHC014 viruses to replicate in human airway cultures, cause patho genesis in vivo and escape current therapeutics, there is a need for both surveillance and improved therapeutics against circulating SARS-like viruses.

Because of the replication of Sas-like coronavirus in the respiratory tract, there is an urgent need for improved treatment.

……

an HIV-based pseudovirus, prepared as previously described, and **examined on HeLa cells (Wuhan Instit ute of Virology) that expressed *ACE2* orthologs**. HeLa cells were grown in minimal essential medium (MEM) (Gibco, CA) supplemented with 10% FCS

Wuhan Virus Institute has a means similar to HIV experiments, as well as

cancer cell strains that can provide expression ACE2. Specific test methods and steps are recorded in detail.

.....

Acknowledgement

Experiments with the **full-length and chimeric SHC014 recombinant viruses** were initiated and performed d before the GOF research funding pause and have been reviewed and approved for continued stud Y by the NIH.

The experiment of the recombinant virus SHC014 was approved by the NIH in the United States.

AUTHOR CONTRIBUTIONS

.....X.-Y.G. performed pseudotyping experiments; K.D. generated structural figures and predictions; E.F .D. generated phylogenetic analysis; R.L.G. completed RNA analysis; S.H.R. provided primary HAE cult ures; A.L. and W.A.M. provided critical monoclonal antibody reagents; **and Z.-L.S. provided SHC014 sp Ike sequences and plasmids.** R.S.B. designed experiments and wrote manuscript.

Shi Zhengli provided the sequence and plasmid of SHC014 (a biological sequence vector that must be used by genetic engineering or viral recombination technology. Plasmid vectors usually carry one or more selective marker genes (such as antibiotic resistance genes) and an artificially synthesized multi-cloned site sequence containing multiple restrictive endonuenuen enzyme identification sites, and most of the non-essential sequences are removed to reduce the molecular weight as much as possible to facilitate recombinant genetic engineering. In other words, without the sequence and plasmids of SHC014 provided by stone, this paper cannot complete all the experiments).

Some readers may still not understand the recombinant genetic engineering operation of the virus. Dr. Xiao La, a virus expert at the National Institutes of Health in the United States, exposed the secret and spread it widely in the industry. Now the virus origin of Wuhan pneumonia virus RaTG13, Shi claimed that it was also found in bats in Yunnan in 2013, but it was not mentioned in the two articles. It was not until the outbreak of the epidemic that it was hurriedly handed over to the gene bank of the United States on January 29, 2020. COVID-19 has more artificially recombinant enzyme suctation sites (restrictive endonulase identification sites) and Furin and ACE2 affinity than RaTG13. Enzymatic site Furin can be easily found in the genetic sequence submitted by the team of Zhang Yongzhen, an adjunct professor of the School of Public Health of Shanghai Fudan University, on January 11. The big news that ACE2 directly attacks human beings can be seen from Shi Zhengli's article in Nature magazine in 2013.

转发一下，NIH萧镭博士的评述

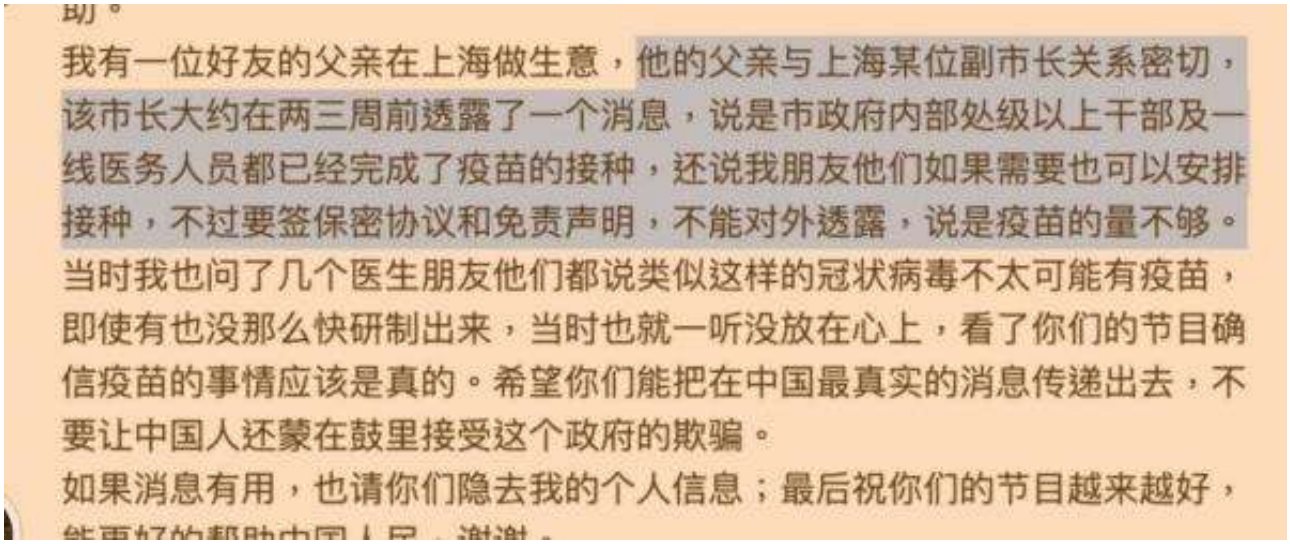
从昨天开始就不断有朋友发给我这段台湾新党党员潘怀宗讲新冠病毒的录像，想听听我的看法。之前因为一直在忙于武汉口罩捐助的事情，根本没时间看。今天问的朋友多了，这里简单谈一下个人的看法。潘讲的关于病毒分类58个家族的说法是根据刚刚在中国科学院科技论文预发布平台上发布的一篇基因测序的论文的内容。该文根本没有经过同行评议，也没有正式发表，属于作者的一家之言，没看出来有任何科学道理！因为目前已知的所有新冠病毒株的序列相似性都在99%以上，根本不存在什么爷爷、父亲和儿子的关系！因为所有病毒都是来自同一个同源病毒！另外，作者提到的蝙蝠的病毒起源株是RaTG13病毒株，这可是全世界仅武汉病毒所石正丽实验室独有的病毒株！她上月对外宣称这是她于2013年在云南大山里蝙蝠洞中发现的，但她7年来从来没有对外报导过，直至这次武汉疫情发生以后，她才在今年1月29日在我和几个朋友的直接逼问下，匆匆把病毒序列交到NIH GenBank。大家现在都不能确定这个病毒本身是真的从蝙蝠身上找到的，还是她自己实验室合成的？有谁听说过哪位科学家发现了这么一个全新的病毒基因后，不抓紧去发到《自然》或《科学》等高分杂志上，而在自己实验室里一放就放了七年，直到武汉出了事儿，才

自己实验室里一放就放了七年，直到武汉出了事儿，才在我们的逼问下匆匆提交的吗？反正我认识的科学家的朋友们不会有人这么做，因为这根本不符合科研常识！再有，RaTG13株和新冠病毒虽然有96.2%的序列相似性，但是新冠病毒是不可能从RaTG13株在自然界中直接进化而成新冠病毒的，这一点最近的发表的科学文献上已经证实了。而且最致命的一点是：两者之间病毒“自然进化”的“中间宿主”动物至今也没有找到！🧐 RaTG13病毒株和新冠病毒的一个重要区别就是不具备furin酶切位点的序列插入。这个序列的插入就是显著增强病毒对人体呼吸道上皮细胞ACE2亲和力的重要改变。直接导致了新冠病毒对人体呼吸道上皮的传染性超过数十倍的增强！而这么精巧的短序列插入的“进化”，在自然界短时间内“自然进化”发生的可能性基本不存在。另外，美国电子烟引起的肺功能变化基本上已经确定是电子烟中伪劣香味调料导致造成的。而且无论给老鼠吸多少烟，短期内是根本不可能引起肺纤维化的。在老鼠中引起肺纤维化模型中只有打Bleomycin才能造模！这是一个常识性的概念，所以，这哥们儿根本不懂！因此，这哥们忽悠的所谓“病毒源头是美国”的结论就更是令人贻笑大方的胡说八道了！非专业的吃瓜群众们切记不要被这种低级的洗地节目带错了方向！👎🤦

Excuse me, Foreign Ministry spokesman Geng Shuang, is the paper published by Shi Zhengli scientific and professional enough? At present, different versions of articles praising the Party are circulating on WeChat,

which means that there are 3,000 casualties of doctors in Hubei, while more than 40,000 medical nurses from other provinces who helped Hubei "none of them were infected", which is amazing. Of course, there will be losses for more than 40,000 medical care, but there will be no shortage of vaccines for the recombinant virus. Shi Zhengli has been preparing for 7 years, and "Nature" is the iron proof!

April 5, 2018 CCTV13 News Domestic Focus, mentioned that the Wuhan virus isolated the novel coronavirus, the ACE2 receptor that can infect humans, and claimed that vaccines and antibodies had been prepared!


助。
我有一位好友的父亲在上海做生意，他的父亲与上海某位副市长关系密切，该市长大约在两三周前透露了一个消息，说是市政府内部处级以上干部及一线医务人员都已经完成了疫苗的接种，还说我朋友他们如果需要也可以安排接种，不过要签保密协议和免责声明，不能对外透露，说是疫苗的量不够。
当时我也问了几个医生朋友他们都说类似这样的冠状病毒不太可能有疫苗，即使有也没那么快研制出来，当时也就一听没放在心上，看了你们的节目确信疫苗的事情应该是真的。希望你们能把在中国最真实的消息传递出去，不要让中国人还蒙在鼓里接受这个政府的欺骗。
如果消息有用，也请你们隐去我的个人信息；最后祝你们的节目越来越好，

The Communist Party of China began to work elaborately again on the WeChat platform. This time, in the name of "overseas Chinese", it began to be washed by Shi Zhengli and the Wuhan virus. The biggest point is to never mention the paper of the most important "Nature" magazine that coaxed the world!!!

全面揭露新冠病毒事件的起源、传播和爆发的真相

原创 宇宙真子 世界华人UFO科学 4天前

【全面揭露新冠病毒事件的起源、传播和爆发的真相】

作者：宇宙真子

本文将全面揭露病毒起源、传播及爆发的来龙去脉（按照时间顺序进行叙述）

一、基因编辑技术与人工合成病毒产生的背景

人类基因组计划、曼哈顿原子弹计划、阿波罗计划并称为三大科学计划。人类基因组计划（英语：Human Genome Project, HGP）是一项规模宏大，跨国跨学科的科学探索工程。其宗旨在于测定组成人类染色体（指单倍体）中所包含的30亿个碱基对组成的核苷酸序列，从而绘制人类基因组图谱，并且辨识其载有的基因及其序列，达到破译人类遗传信息的最终目的。此计划在研究人类过程中建立起来的策略、思想与技术，构成了生命科学领域新的学科——基因组学，可以用于研究微生物、植物及其他动物。

1970年，从基因编辑技术诞生以来，人类就打开了控制物种自然演变的潘多拉魔盒。对人类基因组的研究在70年代已具有一定的雏形，在80年代在许多国家已形成一定规模。

1984年在Utah州的Alta,WhiteR and Mendelsonhn M受美国能源部(DOE）的委托主持召开了一个小型专业会议讨论测定人类整个基因组的DNA序列的意义和前景（Cook Deegan RM,1989)

1985年5月在加州SantaCruz由美国DOE的Sinsheimer RL主持的会议上提出了测定人类基因组全序列的动议，形成了美国能源部的"人类基因组计划"草案。

1986年3月，在新墨西哥州的SantaFe讨论了这一计划的可行性，随后DOE宣布实施这一计划。

1990年正式启动的。美国、英国、法国、德国、日本和我国科学家共同参与了这一预算达30亿美元的人类基因组计划。

2001年人类基因组工作草图的发表（由公共基金资助的国际人类基因组计划和私人

1990年正式启动的。美国、英国、法国、德国、日本和我国科学家共同参与了总预算达30亿美元的人类基因组计划。

2001年人类基因组工作草图的发表（由公共基金资助的国际人类基因组计划和私人企业塞雷拉基因组公司各自独立完成，并分别公开发表）被认为是人类基因组计划成功的里程碑。

二、病毒的来源与中国相关研究的背景

2002年12月中国广东发生SARS即非典事件。

2003年非典大范围爆发，中国开始展开对病毒的研究。

2004年开始，石正丽团队走遍中国28个省市，采集了数千只中华菊头蝠标本，与美国团队合作，进行病毒的基因分析与其他研究。她用了13年时间，走遍中华大地，到处钻山洞抓蝙蝠，最终确定SARS病毒起源于中华蝠。事实上，石正丽进行13年蝙蝠研究的合作伙伴就包括美国生态健康联盟和美国国立卫生研究院。

为何没有石正丽2013和2015年发表在《自然》上的雄文？？？

2008年石正丽团队对蝙蝠的研究有了很多的进步和发现，并发表了相关论文。

2014年以来，CRISPR基因编辑技术带来了一场科学界的革命，全球基因编辑技术发展加快。

2015年01月31日，中国科学院武汉国家生物安全实验室(武汉P4实验室)在武汉建成，标志着中国正式拥有了研究和利用烈性病原体的硬件条件。

2015年01月31日，中国科学院武汉国家生物安全实验室(武汉P4实验室)在武汉建成，标志着中国正式拥有了研究和利用烈性病原体的硬件条件。

2015年10月武汉P4研究人员对蝙蝠携带病毒的研究有了更多进展，包括与艾滋病毒的结合研究。他们曾在美国也有相关的研究和实验。之后石正丽被邀参与在美国的病毒研究与合成计划。

2015年11月9日，石正丽团队在PLosPathgens杂志上发表了一篇名为《一个类似SARS的蝙蝠冠状病毒群显示了在人类中出现的可能性》的论文。内容提到："为了研究循环蝙蝠冠状病毒的出现可能性(即感染人类的可能性)，我们构建了一种嵌合病毒"，这种杂交病毒使我们能够评估这种新的棘突蛋白引起疾病的能力，在此基础上，我们合成了一株具有感染性的全长SHC014重组病毒，并证明了该病毒在体内外的复制能力"。他们通过对云南某一山洞中马蹄蝠连续五年的样品搜集，他们发现了蝙蝠中的SARS相关病毒（SARSr virus）是人SARS病毒祖先的更直接的证据。

2018年美国主导开展了的病毒研究计划。美国研究机构以科学研究的名义对病毒进行提取、培育、编辑、合成等，并在未来打算计划投毒。中国团队在美国的研究中只是提供了一些理论、设计和基础素材。

2019年7月美国生化武器基地德特里克堡因自然灾害原因发生设备故障，之后机构改进了机械设备。由于病毒在管理过程中出现疏忽，8月初生化武器基地德特里克堡被紧急关闭，基地内所有的研究项目全部停止，所有人员将接受全方位的检查，并且美国疾病控制和预防中心CDC剥夺了德特里克堡对包括埃博拉、天花和炭疽等高致命性病毒研究的"特许代理人"许可证。"新冠病毒"泄露，导致大面积感染。最关键的问题是，基地只是做出了病毒，还没有研制出相应的疫苗。这是导致基地被彻底关闭的那个致命

三、病毒爆发的起源与传播的时间线

（一）2019年病毒正式爆发

7月美国生化武器基地德特里克堡发生病毒泄露，并有人感染，之后在周边开始传播与扩散。

德特里克堡基地附近就是美国参加武汉军运会的军人集训基地。由于病毒感染带有隐秘性，许多感染者并不知情。随后病毒不断被无意扩散。病毒泄露导致许多优秀的高手患上了"流感"，无法参赛。即便参赛了，也状态不佳。于是美方只能派遣一些不太优秀的军人参赛。

8月初因为病毒扩散日益严重，受到高层重视，德特里克堡生化基地被迫紧急关闭。

9月病毒开始在美国扩散，并与美国流感同时传播。大量的事实表明，美国去冬今春爆发的所谓流感疫情，其实是流感和新冠肺炎的混合疫情。其中新冠疫情的爆发时间，几乎和武汉的爆发时间同步。但由于美国相对于武汉而言，必定还是地广人稀的。再加上美国对新冠疫情的检测能力不足，这才没有及时显现出来。新冠病毒和流感在短时间内不易被鉴别出来，因为两种患者症状相似，过程相似，CT大同小异，因此被混淆的可能是必然存在的。

10月，美国约翰霍普金斯医学中心主办了一场名为案件201的瘟疫预演，汇集了15名各国政商医届的核心人物，其中包括曾任中情局二把手的艾薇儿·海恩斯，及中国疾控中心主任高福院士。

10月17~27日，第七届世界军人运动会在武汉举行。期间已经有人被感染。

11月病毒开始在武汉传播，同时美国也在蔓延病毒。
12月病毒开始在武汉蔓延。
12月30日，武汉义医吹哨，海鲜市场被封闭。
12月31日第一批国家CDC专家组入场。

（二）2020年病毒开始在全世界大规模爆发

1月病毒开始在中国大范围爆发。
1月3日官方训诫。
1月5日武汉卫健委"没发现人传人"，同日，上海"某中心"上报国家CDC发现武汉不明肺炎人传人病例（上报卫健委的公函一定会转交CDC）。
1月23日10时武汉开始封城。
1月25日蝙蝠病源论公布，但这不是真相。

2月15日，日本名古屋的一对夫妻去夏威夷度假后感染新冠，之后证实和中国无关。同日，日本和歌山有5个人中招，与中国无关。

2月15日科技部出台了《关于加强新冠病毒高等级病毒微生物实验室生物安全管理的指导意见》，要求加强对实验室，特别是对病毒的管理。

2月20日之后，随着疫情在世界各地的迅速蔓延。美国是疫情罪魁祸首的说法开始提调。